citizens. Therefore it is suggested that an authoritative and responsible decision-making body be established in each such area to carry out the program. In a sense this area body might be viewed as a further extension of a concept that has been in place for some time—that health care decisions should be made in local areas by local agencies representing the community to be served. This concept has been evolving through: first the Regional Medical Programs (RMP), then the Comprehensive Health Planning (CHP) agencies, and most recently the Health Systems Agencies (HSA). But to be effective these bodies will need much more clout within their areas than has hitherto been the case. It is essential that *all* those who must carry out a plan also be part of the planning and decision making processes. There needs to be a community or area sense that it is their plan that they are carrying out so what is needed is something more like a "health alliance" which will actually involve all those groups and organizations that are properly concerned with medical and health care services for the area—a responsible agency or authority with real involvement of the providers and "doers" in the area as well as appropriate involvement of consumers and representatives of the underserved. Such an "alliance" could be the authoritative body to provide for the federal "basic" programs and to identify health care problems in the area and what is needed to deal with them, to decide what to do and then work together in the area to get it done.

How might this work? The area "alliance" would receive the capitation funds and make whatever arrangements were necessary to provide for the basic services required by the federal government. Many, perhaps even most, of these services are already in place. Others could be added. Contracts could be entered into by the "alliance" to buy services. Some areas might contract with others for services that could be more easily or more efficiently provided by them. Each area could decide what resources it might wish to add to the basic federal capitation funds in order to improve its health care system beyond the basic requirements for federal capitation. Savings from capitation dollars and funds from other sources could be used for this. In this way competition and cost consciousness could be made an integral part of the delivery system. Existing systems of payment could be used or modified as deemed necessary for efficiency and effectiveness. Federal surveillance would be limited to assuring that the basic services were indeed available to meet the minimum basic standards and to assure the funds were properly accounted for. Professional Standards Review Organizations (PSRO), also based in each area, could monitor quantity, quality and appropriateness of services, and the area "alliance" would evaluate its own programs, cost effectiveness and whatever, just as would any quasi-public business enterprise servicing a given area.

If an idea such as this is to be tried it should be thought through further by persons with expertise this writer does not possess. It should probably also be tried first experimentally in several different kinds of settings to see if it will work. But, if successfully implemented, it should (1) satisfy the political commitment to NHI, (2) put a reasonable and predictable limit on the federal dollars to be allocated to health care services and (3) place the incentives and responsibility for the delivery of health care services upon those who are properly involved in each designated area, which is where it really belongs in our free enterprise system. And this should not be all bad.

—MSMW

Peripheral Nerve Entrapments

YEARS AGO Kopell and Thompson grouped together a diffuse assortment of mononeuropathies, ascribed their cause to localized entrapment and published the now-familiar monograph *Peripheral Entrapment Neuropathies.*[1] The Specialty Conference moderated by Dr. Cracchiola, the proceedings of which appear in this issue of the WESTERN JOURNAL, is a refreshing review of the more common "entrapments." Anatomy, pathophysiology, diagnosis and treatment are outlined. Dr. Campion's succinct discussion of the electrodiagnosis of peripheral neuropathies should prove particularly helpful to the clinician.

The term "entrapment" is, in my opinion, a misnomer. Peripheral nerves are rarely trapped by surrounding tissue, nor is their function likely

to be disrupted because of capture.[2] However, these nerves are sometimes sufficiently compressed by surrounding tissue to cause impairment of nerve function by sheer mechanical force. The median nerve "trapped" in the carpal tunnel then becomes squashed, locally and repeatedly. Pathological specimens, electrophysiological data, and direct observation of nerve deformity at the operating table all confirm that the median nerve can be compressed by the transverse carpal ligament during flexion of the wrist. Similarly, repetitive trauma of the ulnar nerve, its intermittent subluxation across the medial epicondyle and certain anatomic features of the ulnar groove all contribute to produce a "tardy palsy." The pronator teres syndrome, caused by acute or chronic deformity and compression of the median nerve by fibers of the flexor sublimis muscle in the proximal forearm, is another example of a compression mononeuropathy. It may be argued that differences in meaning among the terms compression, mechanical force and entrapment are of merely semantic interest, with little clinical relevance. Yet, when decompression relieves signs and symptoms, compression per se must be acknowledged as the primary etiological factor.

A new clinical concept of peripheral neuropathies has evolved since the time when axoplasmic flow was first recognized. Simply stated, this hypothesis suggests that subclinical lesions in tandem may interfere with nerve function additively, to cause a neuropathy that is clinically indistinguishable from one produced by a single lesion.[3] For example, ulnar nerve compression at the elbow may cause a clinically obvious ulnar neuropathy to appear more rapidly in a patient with diabetic neuropathy than in one who is not suffering from diabetes also. Increasingly sophisticated electrodiagnosis can now identify the separate lesions, and has made precise treatment possible.

Some important facts about peripheral nerves and their responses to injury deserve emphasis here. First, most peripheral nerves have motor and sensory fibers. Hence, compression mononeuropathies cause both motor and sensory signs and symptoms, as a rule. Notable exceptions to the rule are the lateral femoral cutaneous nerve of meralgia paresthetica (a uniquely sensory syndrome), and the deep radial nerve of the posterior interosseus syndrome (exclusively motor). Most other compression neuropathies, including the ubiquitous carpal tunnel syndrome, tardy ulnar palsy and "Saturday night" palsy, involve mixed motor and sensory nerves.

Second, the more proximal the lesion, the wider the distribution of motor and sensory signs and symptoms. In the detection of the sites of nerve injury, it is clear that there is no better analytical tool than the physician's own specific knowledge of peripheral nerve anatomy.

Third, compression of a nerve proximally is not as well tolerated as compression distally. This is because axonal regeneration is more likely to occur when the initial injury is distant from the cell body. Therefore, decompression of a nerve that is compressed distally, rather than proximally, is more apt to be effective.

Fourth, the longer a nerve is compressed, the less likely it is that decompression will result in any improvement.[4]

Transsection or avulsion of a nerve to treat compression neuropathy is rarely necessary. Motor loss following section of a mixed nerve is usually too high a price to pay for transient improvement of a sensory complaint. Occasionally, transsection of the lateral femoral cutaneous nerve for intractable meralgia paresthetica is effective, but more often it is not. "Never cut a nerve" is a time-honored admonition of wise and experienced neurosurgeons. The adage is particularly appropriate in compression neuropathies, for transsection usually changes a simple problem into a more complex one.

Compression mononeuropathy or peripheral entrapment neuropathy should be diagnosed sparingly and by exclusion. Frequently the neuropathy reflects one minor aspect of a larger problem, such as acromegaly, myxedema, rheumatoid disease, amyloidosis and the like. Treatment of the systemic disorder may cure the neuropathy. Similarly, mononeuropathies are often caused by specific lesions strategically placed—a malaligned fracture, a neoplasm within or adjacent to the nerve, or a prior traumatic injury. Sometimes the neuropathy is self-induced, as in meralgia paresthetica caused by pressure from a heavy gun belt, or a carpal tunnel syndrome resulting from the improper use of crutches. Occasionally, neuropathies result from compression by normal body structures, as in the pronator teres syndrome which follows sudden, forceful contraction of the flexor muscles in the forearm, or the carpal tunnel syndrome which occurs in stenographers and jack-

hammer operators. This last group of lesions, the normal anatomy group, is interesting as well as easy to remember. More difficult to diagnose are the widely varying types of lesions that can cause "entrapment" (compression) neuropathies; their identification and successful treatment continue to present a significant challenge.

JULIAN T. HOFF, MD
Department of Neurological Surgery
University of California, San Francisco

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Today's New Physicians

DR. RICHARD G. SWANBERG discusses "The Medical Challenge of Coping with Stress" elsewhere in this issue. The occasion was his address as a graduating medical student at the 23rd Annual Hippocratic Oath Ceremony at the University of California, Los Angeles, School of Medicine, in June of this year. It comes through that for this generation of medical students the stresses of becoming a doctor are much the same as for previous generations, and so is the idealism and grit that it takes to cope and to achieve the goal. The goal is now, as before, to become a quality physician well trained to care for patients, to understand and show compassion for fellow human beings who are under stress, and to "look at the whole patient and not just his disease." And the needs of society are not being overlooked. Three quarters of the graduating class plan to enter one of the primary care specialties "in which a physician is best able to treat the entire patient." All of this should be more than reassuring to legislators and others, in and out of the profession, who have been concerned about the goals and attitudes of today's new physicians. We can welcome these young doctors to our ranks.

—MSMW

Angiotensin Converting Enzyme Levels in Sarcoidosis

SARCOIDOSIS IS A multisystem granulomatous disorder of unknown cause. The disease has a worldwide distribution. In the United States its incidence is estimated at about 30 to 40 per 100,000 of population. Although generally considered a benign disorder, sarcoidosis carries a mortality rate of 5 percent.[1] As a general rule the diagnosis of sarcoidosis is considered secured if all of the following criteria are satisfied: (1) compatible clinical or radiological features, (2) evidence of noncaseating granulomas in one or more organs and (3) negative bacterial and fungal studies of biopsy tissue specimens to rule out other specific causes of granulomatous disease. If all three steps are not carried out the diagnosis of sarcoidosis remains in doubt since clinical or radiological features present too wide a differential diagnosis and histological evidence of noncaseating granulomata may be produced by many bacteria, viruses, fungi and chemicals.[2]

Strict adherence, however, to this three-step diagnostic workup presents two major practical problems. First, the patient, particularly when asymptomatic, may not be willing to have an invasive procedure carried out. Second, biopsy procedures, depending on the site and technique used, carry definite risks of morbidity and even mortality. Consequently for many years the Kveim-Siltzbach test has been identified as another diagnostic test.[3] The test is done by injecting validated suspension of human sarcoidal tissue intradermally in the forearm. In about four to six weeks a tiny nodule appears in about 75 percent of patients with active sarcoidosis. Findings on a biopsy of the nodule show noncaseating granulomas. However, since the Kveim-Siltzbach antigen is not available commercially, the test unfortunately is of no use to practicing physicians.

Recently Lieberman reported a new serological test for sarcoidosis.[4] He found that 83 percent of patients with active sarcoidosis have abnormally high levels of angiotensin converting enzyme (ACE) in the serum. The highest ACE levels were found in persons with bilateral hilar lymphadenopathy and pulmonary infiltrate (stage II sarcoidosis). Resolution of the active disease or